Measuring of the extent of atherosclerotic lesions in the human by means of the assessment of the degree of activation of blood coagulation factor VII

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MEASURING OF THE EXTENT OF ATHEROSCLEROTIC LESIONS IN THE HUMAN BY MEANS OF THE ASSESSMENT OF THE DEGREE OF ACTIVATION OF BLOOD COAGULATION FACTOR VII

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Factor VII is the coagulation factor that is activated by the contents of body cells. The activated factor VII (factor VIIa) in its turn activates factor X. Factor Xa together with factor V forms the prothrombin-converting enzyme, thus being responsible for the formation of thrombin that plays a key role in thrombus formation.

It appears that factor VII activation is not a simple pro-enzyme - enzyme transition. Intermediate activation stages do occur. Native factor VII (factor VIIn) is converted into a state with enhanced activity (factor VIIe) that is transformed in fully activated factor VII (factor VIIa) which is quickly inactivated (factor VIIi).

The products of the reaction sequence
factor VIIn - factor VIIe - factor VIIa - factor VIIi
each have their own half-life in the circulation. We developed methods to assess the forms n, e, and a separately.

We tested the hypothesis that cell wall lesions cause a partial activation of factor VII. To this end we assessed the form and degree of activation of factor VII in groups of patients known to have extensive atheromatous degradation of the intima of major arteries. One control group existed of normals, another of patients with generalized carcinomatous disorders.
It appeared that the difference between cell wall lesions by atheromatous degeneration and carcinomatous infiltration is reflected in the state of activation of factor VII.

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